testing for central scotoma and with a light should, however, frequently aid in the exact diagnosis. Mine has certainly been an ideal patient making the perimetric test rather of the nature of an exact physiologic experiment. Still I rather wonder that in no contribution heretofore, as far as I am aware, the fact has been noted that, as a matter of course, the scotoma in the typical segment-shaped hemorrhage will be inverted to the form of its anatomical substratum** Haab (l. c.) refers to his sixth patient as being very intelligent and a good observer, hence it had been possible to ascertain that she had a scotoma that corresponded in form "exactly" to the hemorrhage. Only Obermaier has it more directly, in parts viz., that the perimeteric examination revealed for either eye a central scotoma with sharp "arch-like upper" border, there being a relative scotoma adjoining the absolute scotoma, downwards, corresponding to the upper transparent layer of fluid of the ophthalmoscopic picture.

In conclusion I wish to refer briefly to a few anatomical data that seem to be helpful for an understanding of the way in which the hemorrhage takes places. According to Marcus Gunn (quoted by Haab, l. c.) the hyaloidea is not as firmly attached to the retina in the macula as elsewhere. Rather plausible further is Dimmer's (l. c.) explanation. In the macular region according to Dogiel, Mueller's fibres frequently split into 2 or 3, some distance from the inner surface of the retina. The inner ends of these secondary fibres then, as they form the margo limitans, being much finer than the conical endings of Mueller's fibres elsewhere render it easier for a hemorrhage to break through at the macula.

REFERENCES.

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2 Graefe-Saemisch V. 1st ed.
3 Traite complet d'ophth. Tab. IV.
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THE BACTERIOLOGY AND PATHOLOGY OF PLAGUE.

(With the Demonstration of Gross and Microscopic Specimens.*)

By WM. B. WHERRY, M. D., San Francisco, Bacteriologist to the San Francisco Board of Health.

It will be impossible to give a full account of this subject in the allotted time so I will simply review a few facts which physicians and epidemiologists must always have in mind.

The exhibit of microscopic specimens should impress upon your minds the morphology, size and staining peculiarities of *Bacillus pestis*. I would call your particular attention to the preparation showing the "coccoid" form of *Bacillus pestis,* as these very characteristic forms are often encountered in the tissues of both rats and human beings. The exact manner in which these coccoid forms are produced in the animal body is still an open question. They are undoubtedly involution forms but viable for they always produce acute plague in experimental animals. I accidentally discovered that they could be produced in guinea pigs by the injection of the involution forms of *Bacillus pestis* grown on salt agar. It is probable that the salt content of the tissues has something to do with their production as shown by ash determinations kindly made for me by Mr. Beaver and Mr. Hyde of the Chemical Laboratory of the San Francisco Board of Health. The spleen and gland from a rat showing these coccoid forms and from a plague rat showing typical bipolar bacilli were examined and the tissues of the former contained about 0.5 per cent more solids by weight.

The bacteriologic identification of the plague bacillus, apart from animal experiments, depends chiefly upon two tests: The production of typical involution forms on salt agar containing two or three per cent of pure sodium chlorid and upon the production of typical salactite growths in oiled bouilon.

Viability Outside the Animal Body—Like many other nonspored organisms *Bacillus pestis* is very susceptible to the destructive agencies of nature. Exposure to direct sunlight (on coverglasses) kills it in an hour. Dried up in organs, on clothing, or in soil it dies in 5 to 15 days according to temperature conditions. Exposure to 55°C. (moist heat) for 15 minutes is fatal. One to 1000 bichlorid of mercury kills it immediately; and 5% carbolic in one minute. Like many other micro-organisms it may be frozen to many degrees below zero (Centigrade) without injury.

Pathogenicity—The plague bacillus is pathogenic to a number of animals besides man. In considering the susceptibility of animals it will be well to separate in your minds those that have been observed to acquire the disease *naturally* from those that may be infected *artificially,* e. g. by the injection of massive doses.

Among the rodents plague naturally occurs among the Norway rats (*M. Rattus*); the Egyptian roof rats (*M. rattus alexandrinus*); mice (*M. musculus*); the *Nesokia Bengalensis*—small bandicoots in India, closely resembling the Norway rat; the tarbagan (*Arctomys Bobac*) a rodent, related to the marmot, which inhabits the mountains in East Siberia and is also found in the Himalayas where epidemics of plague are said to decimate them. (Clemow. *Joun. Trop. Med.,* Feb. 1900.)

The observation of Liston that epizootic plague may run through guinea pigs gave the last Indian

** The image of an outer object upon the retina is an inverted one, and the object is seen erect by a psychical process. In the language of Helmholtz we see the sun and stars an den Himmel not an dem Himmel, on to or into, not upon or in the sky. All retinal sensations are projected outwardly to the opposite side of the visual field, as is easily demonstrated by the simple experiment of pressure phosphene. If one presses the globe at the nasal side its bearer will experience the sensation of light (or dark) on the temporal side. It follows that entoptic phenomena originating behind the nodal point (in the sense of the reduced Donders' eye) are experienced outside of the eye inverted to their cause, and speaking teleologically there would seem to be no reason for a psychical act of reversing. Clinically the matter has not been used. I have tried in suitable cases, e. g. of grotesque vitreous opacities, to find in the outlines of the positive scotoma, as drawn by the patient, the contour of the ophthalmoscopic picture reversed, but have failed—evidently, the shadow from the formations as anticipated upon the retina from the frontal aspect is still largely modified by their corporeal structure posteriorly.

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commission one of its most valuable means of studying the manner in which plague spreads through an Indian village. According to J. A. Thompson marsupials acquire the disease in Australia. (*Jour. Hyg.,* 1906, 6, 549.) Several species of monkeys naturally acquire it. Cats may develop a chronic form with cervical buboes. Rabbits and squirrels are susceptible to inoculation but I am unaware of any authentic observation of a natural epizootic among them. Recently (1907) Biffi of the University of Lima has shown that the llama is susceptible to inoculation and dies in eight days with characteristic lesions.

Wilm (1896-7) claimed to have transmitted plague to many of man's domestic animals by feeding experiments; and in 1903 Simpson made a series of experiments to show that pigs, hens, calves, ducks, geese and sheep can be infected by feeding with the organs of animals dead of the disease. An analysis of the work of these experimenters shows that they failed to distinguish between the hemorrhagic septicemias of animals and plague.

Bannerman repeated Simpson's experiments in India (1904) and Hill in Natal (1904) and both failed entirely to convey plague to any of the domestic animals.

Mode by which the Plague Bacillus Gains Entrance into the Animal Body—Infection in man occasionally occurs through inhalation, resulting in primary plague pneumonia. Still more rarely by rubbing infective material through an occupation or an accidental wound. But most important of all is the rubbing of infective material into the atrium afforded by a flea bite. It will be necessary here to briefly summarize the findings of the last Indian commission on this point. (*Jour. Hyg.,* 1907, 7, No. 3.)

1. Both male and female rat fleas may take up as many as 5000 bacilli from the peripheral circulation of a plague-infected rat.

2. The bacilli multiply within the flea and such a flea may pass out large numbers of virulent germs with its feces during ten to fifteen days after infestation.

3. Fleas have a well recognized habit of defecating while sucking blood and this, in the case of a plague-infested rat flea, results in the deposition of infective material near the site of its bite.

4. A flea bite has been shown experimentally to be a sufficient atrium of infection.

While the majority of the Indian commission's experiments were performed with the rat flea *Loemopsylla cheopis,* they also made two experiments with another rat flea *Ceratophyllus fasciatus* and both of these were successful. All of twenty-seven experiments with *Pulex felis,* the cat flea, failed. Only three out of thirty-seven experiments with the human flea (*Pulex irritans*) were successful and this low percentage was explained by the fact that human fleas do not thrive well upon rats and guinea pigs.

Pathology—At the necropsy table we make distinction between three main types of the disease in man.

1st. Primary bubonic plague; most frequent of all.

2nd. Primary septicemic plague.

3rd. Primary pneumonic plague.

When plague bacilli gain entrance into an infection atrium of the skin, very rarely they first localize there giving rise to a primary plague pustule of the skin which when fully developed closely resembles an anthrax carbuncle. This is shown very well by the specimen on exhibit from the case of Max P. E.

In the vast majority of cases, however, the bacilli are carried directly by the lymph stream to some of the superficial regional lymphatic glands. If their progress is arrested here, the further multiplication of the bacilli gives rise to the *primary bubo* which is characterized by an intensely hemorrhagic inflammatory process with a consequent fusing of adjacent glands, and the pouring out of a plastic serogelatinous exudate into the peri-glandular tissues.

When this invasion of the glandular tissues is of a comparatively mild grade and the further progress of the bacilli is arrested we speak of the case as one of *pestis minor,* or clinically, if accompanied by few symptoms, as one of *mild* or *ambulatory plague.*

However, if the bacilli in the glands of such a bubonic case pass the lymphatic filters the patient suffers from a *secondary septicemia* with or without a *secondary plague pneumonia.*

When plague bacilli on their way from an infection atrium are not arrested by the regional glands and make a rapid entrance into the circulation they give rise to *primary septicemic plague.*

When infection is acquired through close association with a primary or secondary pneumonic case, the type produced is usually one of *primary plague pneumonia.*

The anatomical changes may be briefly summarized as follows:

The postmortem rigidity and levidity are usually marked. Small petechial or ecchymotic hemorrhages into the cutaneous tissues or under the serous membranes, particularly of the lungs, heart, kidneys, cerebral and spinal meninges may be found. Submucous hemorrhages may also be present in the stomach, intestines and bladder. These submucous and subserous hemorrhages are very well shown by the kidney and stomach on exhibition.

However, it should be remembered that while such petechiæ are often present in primary and secondary septicemic plague, they also occur in other diseases,—notably in cerebrospinal meningitis, tick fever, typhoid and typhus fevers, beri-beri, and in some cases of severe streptococcus, pneumococcus and *bacillus mucosus capsulatus* infections. The primary bubo has been described. Many of the lymphatic glands may be infected through the blood stream giving rise to buboes of the second order. These are not as congested nor as hemorrhagic as the primary bubo and the glands in a group are not fused together.

The internal organs show the usual changes accompanying many acute bacterial infections, such

as: splenic tumor; acute nephritis; congestion of the liver; myocarditis, often with dilatation; congestion of the brain and cord; and hyperemia and swelling of the bone marrow. In pneumonic plague the lungs present the picture of a typical confluent lobular pneumonia, are blackish red and very hemorrhagic on section and microscopically show enormous numbers of typical bi-polar staining bacilli.

Types of the Disease in Rats—Rats may have either acute or chronic plague. In acute rat plague the most important signs are: the presence of a typical bubo; of a liver showing small yellowish foci of necrosis; and hydrothorax. Marked subcutaneous congestion and noticeable enlargement of the spleen may or may not be present.

Hitherto, with the exception of Hunter who described an intestinal form of chronic plague in rats, most observers have found the chronic lesions in the form of abscesses either in the spleen or connected with the spleen or a lymphatic gland. In our experience with a few cases of chronic plague in rats in San Francisco the lesions have always been in relation to the spleen or a gland.

THE OPTHALMO-TUBERCULIN REACTION.*

By EDWARD F. GLASER, M. D., San Francisco.

This is a brief report on the limited observation of in all 39 cases, some from the Fruit and Flower Mission Clinics and a few private office cases. We have used chiefly Vicario's, the French Solution of Tuberculin, it being self evident important to have a reliable and standard preparation.

The Vicario's is a 1% watery solution or more properly speaking suspension of tuberculin, i. e. briefly, the tubercle bacilli or fragments of them, destroyed by heat, precipitated and purified by alcohol and dissolved or suspended in a watery solution.

Vicario's 1% solution is sometimes called the No. 2 solution in distinction from a No. 1 solution which is ½% solution recommended by Calmette, sent out from the Pasteur Institute and put up by Poulenc Freres. The tuberculin can also be obtained dry in small cells sent out from the Lille Pasteur Institute, to which it is directed to add a certain amount of water to make a ½% solution and half that amount to make a 1% solution. Parke, Davis & Co., send out the tuberculin in the form of compressed tablets which are directed to be dissolved in a certain amount of distilled water.

The technic is very simple. With the patient's head tipped slightly backwards, a drop is instilled into one eye alone (the other being used as a control); preferably instilled at the inner angle of the eye into the conjunctival sac or on the caruncle and the lids kept apart for a few minutes to avoid the expulsion of the drop by the involuntary closing of the lids. Where one has several patients to try it on at one time, a convenient and economical method is to draw up the requisite number of drops from the containing ampulle or little bottle into a hypodermic syringe and then inject as it were, exactly a drop upon the conjunctiva of the eye.

As stated before the 1% solution was generally used except in patients under 15, when the ½% was employed, although as an experiment the 1% solution was used on a non-tuberculous child 4 years old without producing any redness or irritation and also the 1% solution in a tuberculous child of 6 years produced no excessive reaction, and in the case of a suspect 11 years of age in which one drop of the ½% solution was negative, a week afterwards two drops of the 1% solution instilled into the other eye produced also absolutely negative results.

Many recommend the use of the ½% solution in all cases and then in the absence of a reaction the instillation later of the 1% in the other eye, but as with care to the eye no troublesome conjunctivitis has appeared, it would seem better in adults to use the 1% solution, rather than to have the doubt which might follow the nonreaction to a ½% solution.

For accurate observation, the condition of the ocular and palpebral conjunctiva and of the caruncle should be carefully noticed before the instillation. In our list of cases the earliest showing of the reaction was about 2 hours, between 5 and 6 hours after the instillation being the average; about 12 hours was the latest appearance, although the literature speaks of appearances 48 hours after the instillation. The presence of the reaction is indicated by a shiny, bright redness over the caruncle and the lower palpebral conjunctiva and generally an injection of the lower and inner quadrants of the ocular conjunctiva, showing there as a network or tracery of blood vessels. A couple of hours after the appearance of this congestion, on drawing down the lower lid, there may be found strings of grayish fibrinous exudate and over the caruncle this, with muco pus, may form what almost looks like a membrane. The caruncle is generally slightly swollen, and sometimes the lower lid. The subjective symptoms may be almost nothing as in one case in which the patient gave a most typical reaction yet said that he felt no difference, up to another case with only a doubtful reaction, who complained of such distress that he said that he was afraid he might lose his eye; but surely his pain must have been imaginary.

Generally the subjective symptoms are lacrymation, some photophobia, slight smarting or scratching of the lower lid as though there might be a little dust in the eye, the lids stick together in the morning, and, with abundant secretion, some slight disturbance of vision. But as a rule the subjective symptoms are not annoying. The after treatment consists in cautioning the patient about rubbing the eye, keeping out of wind and dust and use of boric acid solution. In no case have we bandaged the eye. The reaction disappears in from 2 to 10 days. The filaments of fibrinous exudate and muco pus are not found much after the first 24 hours of their appearance. A summary of the 39 cases shows 7 clinically tubercular cases gave positive reaction; 8 suspects gave positive reaction; 2 luetic gave positive reaction; 2 clinically tubercular gave doubtful reaction; 2 suspects gave doubtful reaction; 9 suspects gave nega-

* Read before the San Francisco Society of Eye, Ear, Nose and Throat Surgeons.